University of Alberta

Assessment of Fine Motor Control in Patients with Occupation-Related Lateral Epicondylitis

by

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DEDICATION

To my family ... whose love and support made this research project possible

ABSTRACT

Lateral epicondylitis (LE) is a common overuse injury related to a mechanical overload of the wrist extensors' origin, however, some patients also complain of clumsiness suggesting a possible motor control problem. The purpose of this study was to examine for differences in fine motor control ability between subjects with LE and age, gender, and hand dominance matched control subjects (n = 28) using the Purdue Pegboard Test (PPT) and the Complete Manual Dexterity Test (CMDT).

The LE group demonstrated a significant decrease in fine motor control ability on both measures, compared with the control group on both the PPT, $F(1,52) = 9.98$, $p < .005$, and the CMDT, $F(1,52) = 18.11$, $p < .001$. This suggests that tests of fine motor control should considered in the assessment of clients with lateral epicondylitis. The mechanism related to the deficit is unknown and warrants further research.

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LIST OF ABBREVIATIONS

C	Control Group
CARD	Cumulative Activity Related Disorder
CMDT	Complete Manual Dexterity Test
Dom	Dominant Limb
ECRB	Extensor carpi radialis brevis
ECRL	Extensor carpi radialis longus
ECU	Extensor carpi ulnaris
EDC	Extensor digitorum communis
EMG	Electromyography
Non-dom	Non-dominant limb
LE	Lateral Epicondylitis Group
LTP	Long term potentiation
LTD	Long term depression
MEG	Magnetoencephalography
PPT	Purdue Pegboard Test
TMS	Transcranial Magnetic Stimulation

INTRODUCTION

Lateral epicondylitis of the elbow (LE) is a form of soft tissue injury, often referred to as tendonitis or tendinitis, which involves the tendinous origin of the wrist and finger extensors (Nirschl & Ashman, 2003). LE is a common occupational injury, and is part of a larger family of upper extremity soft tissue disorders called "repetitive strain injuries" (Ashbury, 1995; Yassi, Sprout, & Tate, 1996). The "overuse" or "biomechanical" model of tendonitis has focused primarily on the repetitive mechanical overloading of the tendon beyond its adaptive and reparative capacity as the primary cause of signs and symptoms (Jarvinen et al., 1997; Melborn, 1998; Moore, 2002). Repeated loading of the muscle-tendon unit is thought to cause fatigue-type tears which may fail to repair if the load is continued, or if other physiological factors are involved. Damage may range from micro tears within the collagen fibrils to total structural failure and rupture of the tendon. (Butler, Grood, Noyes, & Zernicke, 1978; Knorzer et al., 1986; Michna, 1987). Most problems appear to occur at the muscle-tendon junction. A subsequent local inflammatory response was assumed to explain the client's symptoms of pain. More recently, however, the view of tendon injuries such as LE as inflammatory disorders has been challenged since few or no signs of inflammation are typically present (Barr & Barbe, 2002; Galliani et al., 2002; Khan, Cook, Taunton, & Bonar, 2000; Maffulli, Wong, & Almekinders, 2003). The term "tendinosis" is now preferred, signalling degenerative changes within the tendon (Khan, Cook, Taunton et al., 2000).

As inflammatory mediators do not appear to be responsible for the pain associated with chronic tendon problems, alternative explanations for the persistent symptoms are being explored. Some potential pain mechanisms include: pain arising from mechanical disruption of the collagen fibrils within the tendon (Gotoh, Hamada, & Yamakawa, 1998; Khan, Cook, Maffulli, & Kannus, 2000), changes in the normal tendon regulatory control mechanisms due to effect of neuropeptides (Hart et al., 2005; Ljung, Alfredson, & Forsgren, 2004), or problems in the pain regulatory system such as peripheral or central sensitization (Bolay & Moskowitz, 2002; Flor, 2003; Giamberadino, 2003). The effect of repetitive movement on the somatosensory cortex also has interested some researchers. Repetitive movement patterns have been associated with the development of peripheral repetitive strain injuries, such as lateral epicondylitis (Barr & Barbe, 2002; Jarvinen M., Jozsa, Kannus, Jarvinen T., Kvist & Leadbetter, 1997; Luopajarvi, Kuorinka, Virolainen, & Holmberg, 1979). Highly repetitive movement patterns have also been shown to produce cortical changes which may lead to impaired motor performance (N. Byl et al., 1996; Byl & Melnick, 1997; Byl et al., 1997; Byl, Merzenich, & Jenkins, 1996). Changes in movement strategies and altered muscle recruitment patterns, whether due to pain or central alterations in motor control, may result in increased loading of the already compromised structures within the muscles and tendon attachments (Barr & Clark, 2004; Byl, 2004; Byl & McKenzie, 2000; Ervilha, Arendt-Nielsen, Duarte, & Graven-Nielsen, 2004). Empirically, this author has noted that patients in the clinical setting will often describe a feeling of "clumsiness" associated with their condition of lateral epicondylitis, suggesting the possible existence of a fine motor control problem. Failure to recognize or address problems with fine motor control may be

one explanation for the persistence of difficult cases of lateral epicondylitis that are resistant to existing treatment approaches, which are primarily mechanically-based and aimed at the peripheral musculotendinous structures.

Definition of Lateral Epicondylitis

Lateral epicondylitis is a common condition which manifests itself clinically as pain usually localized over the lateral aspect of the elbow and upper forearm during or following activities involving gripping. The layperson's term, *tennis elbow*, is commonly used to describe lateral elbow pain and first appeared in the literature in 1882, to describe a painful condition of the elbow associated with English lawn tennis (Morris, 1882). It has since been estimated that only five to ten percent patients with tennis elbow actually play tennis (Assendelft, Hay, Adshead, & Bouter, 1996). Workers in occupations involving repetitive hand-intensive work, however, appear particularly at risk to develop lateral epicondylitis (Chiang et al., 1993; Dimberg et al., 1989; Kivi, 1984; Kurppa, Viikari-Juntura, Kuosma, Huuskonen, & Kivi, 1991; Rayan, 2002).

The term "epicondylitis" was used by Coues (1914) and Cyriax (1978) to describe lateral elbow pain, under the assumption that the presence of pain indicated there was an inflammatory pathology present. Histological studies, however, have demonstrated little evidence of inflammation in chronic cases of lateral epicondylitis (Barr & Barbe, 2002; Green et al., 2002b; Maffulli et al., 2003). Degenerative changes in the tendons of the lateral epicondyle are often observed on surgical biopsy, therefore, the current recommendation is that chronic tendon disorders be referred to by the term

tendonosis (Khan, Cook, Taunton et al., 2000). Despite this recommendation, and the confusion in the terminology used to describe lateral elbow pain, the term lateral epicondylitis will be used throughout this paper since this is the term that has been generally used in the literature and in practice.

BACKGROUND

The literature was reviewed to better understand the current scientific understanding of the relevant anatomy, incidence, diagnosis, pathology and treatment of lateral epicondylitis. Further, background information on fine motor control, use-dependent neuroplasticity, and the effect of pain on motor control, was explored to gain an understanding of the sensorimotor changes associated with repetitive movement. The following review was largely limited to these purposes.

Anatomy and Biomechanics

The dorsal forearm muscles originate primarily from the lateral aspect of the lower humerus, and both extend and deviate the wrist. Moore (2002) has provided an excellent summary of the functional anatomy, as it relates to lateral epicondylitis, which is summarized below.

The three primary muscles involved as possible sources of lateral elbow pain include the extensor carpi radialis longus (ECRL), the extensor carpi radialis brevis (ECRB), and the extensor carpi ulnaris (ECU). The ECRL muscle originates from the supracondylar ridge and lateral intermuscular septum, and occasionally the upper portion of the lateral epicondyle, and inserts on the dorsal surface of the base of the

third metacarpal. The ECRB muscle originates from the lateral epicondyle, the lateral intermuscular septum and the common extensor tendon, and is often fused with the ECRL muscle. Finally, the ECU muscle originates from the lower pole of the lateral epicondyle as a portion of the common extensor tendon and inserts on the medial side of the base of the fifth metacarpal. All three muscles extend the wrist when contracted, however, due to the biomechanical angle of pull the ECRL and ECRB muscles will also contribute to radial deviation of the wrist, while ECU will assist in ulnar deviation of the wrist. The extensor digitorum communis muscle (EDC), which is the primary muscle for extension of the fingers, lies just superficial to the tendinous origin of the ECRB muscle and their tendons blend together, making it difficult to separate them by palpation. Another possible source of lateral elbow pain is the lateral collateral ligament which functionally separates the lateral aspect of the elbow into anterior and posterior halves. This ligament is the primary lateral stabilizer of the elbow, and accidental disruption of the lateral collateral ligament during lateral release surgery has been implicated as a reason for failed surgical results with lateral epicondylitis (Morrey, 1992).

Snijders, Volkers, Mechelse, & Vleeming (1987) described a mathematical model of the biomechanical forces involved during pinching and grasping, associated with lateral epicondylitis. The sum of all moments and forces around the wrist must be in equilibrium to maintain a stable wrist during grasping. The force of the contraction of the wrist and finger flexor muscles during resisted grasping would tend to flex the wrist if there were not an equal and opposite force exerted by the extensor muscles of the wrist and fingers. Thus, any increased pinching, grasping and gripping activities

will require increased wrist extensor activity to stabilize the wrist and optimize the length-tension relationship of the finger flexors. The "overuse" of the wrist extensor muscles during repetitive grasping and pinching movements helps explain why a decrease in maximal and/or pain free grip is often used as an measure of severity of lateral epicondylitis, and as a measure of progress with treatment (Pienimaki, Tarvainen, Siira, Malmivaara, & Vanharanta, 2002; Smidt, van der Windt, Assendelft, Mourits et al., 2002).

Etiology

Tendon injuries can arise from multiple mechanisms including: traumatic laceration, acute rupture during excessive loading, direct physical trauma such as a contusion; or can begin gradually through a more insidious pattern of overuse. Epidemiological studies and experimental research have demonstrated an association between chronic repetitive strain and the development of musculotendinous injuries, however, the relationship between the degree of exposure and tissue pathology is still largely unknown (Armstrong, Ogilvie, & Schwane, 1983; Barr & Barbe, 2002; Jarvinen et al., 1997; Stauber, Smith, Miller, & Stauber, 2000). In clinical practice, the most common history for lateral epicondylitis is a gradual onset of symptoms, and direct trauma very rarely appears to be a cause (Boyer & Hastings, 1999). It is this non-traumatic lateral epicondylitis of overuse origin which was the focus of this study.

Lateral epicondylitis often appears related to occupations with highly repetitive physical job tasks involving extensive hand and forearm muscles (Luopajarvi, Kuorinka, Virolainen, & Holmberg, 1979; Roto & Kivi, 1984). Specific risk factors

thought to be associated to the development of lateral epicondylitis are: movements which require repeated resisted pronation and supination of the forearm, repetitive or forceful grasping, frequent use of pinch grip, and lifting with the palm in a downward position (Coonrad & Hooper, 1973). General risk factors for repetitive strain injuries may include: biomechanical factors such as working in awkward positions or faulty posture, physical factors such as previous injuries, cold, and lack of physical activity, and psychosocial factors at the workplace such as stress and job dissatisfaction (Burton, Polatin, & Gatchel, 1997; Gilbert et al., 1997). It is difficult to 'prove' causation of many repetitive strain injuries due to the difficulty in objectively quantifying the loading of muscles at work, as well as the lack of a standardized method of movement analysis (Harris & Harber, 1997; Kay, 2003; Norregaard, Jacobsen, & Kristensen, 1999). It is most likely that there is a complex interaction between multiple factors of individual susceptibility and physical workload, which manifests itself as a repetitive strain injury (Leclerc, Landre, Chastang, Niedhammer, & Roquelaure, 2001; Viikari-Juntura, 1998).

Epidemiology and Natural History

Lateral epicondylitis appears rather prevalent in the general population. Epidemiological studies in Sweden have suggested a incidence of approximately 4 to 7 percent of the general population, with a peak occurring between the ages of 35 and 54 years (Assendelft et al., 1996). Lateral epicondylitis appears to occur equally in men and women, but is more common in Caucasians (Coonrad & Hooper, 1973). Lateral epicondylitis, as previously mentioned, appears to be a common work-related overuse injury. A five year Swedish study by Kivi (1984), which explored the

incidence of occupation-related upper limb disorders, reported 24 percent of the 3,090 cases he examined were diagnosed as lateral epicondylitis. The reported incidence of lateral epicondylitis in industrial workers in the United States is 7.4 percent (Dimberg, 1987). Workers in occupation and industries involving repetitive or vigorous hand use appear particularly susceptible to developing a repetitive strain injury, for example, meat packing, poultry processing, cashiers, computer keyboarding, welding, painting, auto repair, driving, musicians, assembly line workers, athletes, mail sorting, dentists, and other occupations which involve repetitive movement in awkward postures (Gilbert et al., 1997). Workers in time-pressured, assembly-line-type occupations seem particularly prone to developing lateral epicondylitis. For example, the incidence of lateral epicondylitis in 207 employees who were examined in a fish processing plant was 14.5 percent (Chiang et al., 1993).

In Alberta, out of a total of 2,186 Workers' Compensation claims for tendonitis in 2003, 842 of these claims were diagnosed as lateral and medial epicondylitis (Personal Communication, Rita Yim, Statistical Services WCB AB, Jan 6, 2004). The reported incidence in 2003, also was greater than any of the previous three years. Although the Workers' Compensation Board does not differentiate between medial and lateral epicondylitis, the latter is far more common. Medial epicondylitis has a reported incidence rate of 1.5 percent among workers, versus the nearly 20 percent for lateral epicondylitis (Descatha, Leclerc, Chastang, & Roquelaure, 2003). Thus, the majority of the 842 claims are assumed to be lateral epicondylitis. These claims were of non-traumatic origin with a gradual progressive onset of symptoms, while only 67

claims in 2003 were diagnosed as a traumatic epicondylitis with a history of a specific incident. This finding supports the general view that the majority of cases of epicondylitis have a gradual onset, presumably due to overuse rather than an acute traumatic injury.

Despite the high incidence of lateral epicondylitis, there is little consensus on the best treatment approach for this condition, and there is minimal research to guide the clinician (Bowen et al., 2001; Labelle et al., 1992; Smidt et al., 2003). Lateral epicondylitis has generally been considered as a self-limiting condition that will resolve gradually with adequate rest and time, and resolution rate of 70 to 80 percent at one year has been reported (Boyer & Hastings, 1999; Burgess, 1990; Haahr & Andersen, 2003). There appears, however, to be a smaller subpopulation of patients with chronic or recurrent problems, and increased disability. In this group of patients the condition can persist for as long as to 48 months, and clinically these patients present a significant treatment challenge (Haahr & Andersen, 2003; Murtagh, 1988; Verhaar, 1994).

Pathological Process

The term lateral epicondylitis implies an inflammatory cause for the pain; however, little histopathological evidence of an acute or chronic inflammatory process exists (Barr & Barbe, 2002; Galliani et al., 2002; Khan, Cook, Taunton et al., 2000; Maffulli et al., 2003). These findings may explain the often mixed response to treatments aimed solely at reducing inflammation, such as anti-inflammatory medication or steroid injections (Assendelft et al., 1996; Green et al., 2002b; Smidt, van der Windt,

Assendelft, Deville et al., 2002). It is now appreciated by most researchers and clinicians that the traditional soft tissue healing model with a prolonged inflammatory response does not fully explain the pathology involved in tendonitis

The first detailed histopathological investigation into lateral epicondylitis was by Goldie (1964) who determined that degenerative changes near the attachment of the extensor carpi radialis brevis muscle was the primary pathology associated with this condition; a finding confirmed by several other authors (Nirschl & Pettrone, 1979; Potter et al., 1995). This degenerative process has been termed angiofibroblastic degeneration, and is characterized by an invasion of the tendon by atypical fibroblasts and vascular granulation tissue. In more advanced stages of tendinosis, angiofibroblastic degeneration can also spread into the normal surrounding supporting tissue (Kraushaar & Nirschl, 1999; Nirschl, 1992; Nirschl & Pettrone, 1979; Regan, Wold, Coonrad, & Morrey, 1992). Evidence of degenerative tendon changes with chronic lateral epicondylitis also has been supported by investigation comparing the normal histological features of the common extensor origin of subjects without known lateral epicondylitis to those with the condition. During a single blinded histopathological study of 12 cadavers with no history of lateral epicondylitis, a trained pathologist was unable to identify any histological changes in all of the twelve control specimens. In contrast, abnormal histopathological features were identified in all eleven surgical specimens, including vascular proliferation and focal hyaline degeneration (Regan et al., 1992). These findings suggest that the degenerative changes seen in pathological specimens do not simply represent a normal degenerative aging process. One possible weakness of surgical biopsy studies,

however, is that these specimens have been taken only from chronic cases, who have usually undergone extensive conservative treatment which may have altered the histological tissue characteristics (Boyer & Hastings, 1999). Also, these surgical cases represent only a very small sample of the overall population of lateral epicondylitis (< 5%) and may not be representative of the larger population (Buchbinder, Green, Bell et al., 2002). While angiofibroblastic degenerative changes are well documented in chronic tendon injuries, there is still controversy about the pathological process across the time course of tendonitis. Is it possible that inflammation may be present in the early stages of acute tendonitis followed by repair and/or fibroblastic scarring (Barr and Barbe, 2002; Barr, Barbe & Clark, 2004, Uchio et al., 2002; Waugh, 2005).

Pain Mechanisms

Since inflammatory mediators do not appear to account for the pain associated with overuse tendon injuries, other theories have emerged. One simple theory on the pain mechanism involved in tendonitis is that there is a separation or tearing of collagen fibers; analogous to the pain associated with an acute grade I or II ligament injury. This view has been refuted by some authors, however, as it has been observed that there is often minimal pain with complete excision of collagen following a surgical excision for a patellar tendon allograft anterior cruciate ligament reconstruction, or with a complete rotator cuff tear, both which involve serious disruptions of collagen tissue (Gotoh, Hamada, & Yamakawa, 1998; Khan, Cook, Maffulli, & Kannus, 2000).

More recently, it has been proposed that the pain associated with tendon injury involves a biochemical-neurological mechanism. This model proposes that neuropeptides, either acting directly on free nerve endings or indirectly via mast cells, are involved in tendon regulatory control, and that this control may become dysfunctional and contribute to either inflammation or failure to repair tendon damage (Ljung, Alfredson, Forsgren, 2004; Hart et al, 2005). Functional reorganization within the somatosensory and motor system has been implicated in the persistence of chronic musculoskeletal pain (Flor, 2003; Giamberardino, 2003). As with other chronically painful conditions, it is possible that the persistence of the pain associated with lateral epicondylitis could lead to problems in the pain regulatory system resulting in a neuropathic chronic pain syndrome (Bolay & Moskowitz, 2002; Harden, 2005).

Diagnostic Criteria for Lateral Epicondyltitis

Lateral epicondylitis is generally diagnosed on the basis of the patient history and clinical examination including the use of manual provocation tests, and the exclusion of other factors such as cervical radiculopathy or elbow joint pathology. Patients often describe pain over the lateral aspect of the elbow provoked by grasping with an extended elbow; and the grip strength of the affected limb is often diminished when compared bilaterally. A recently published multivariate analysis of the diagnostic variables suggested that pain on loading the common extensor origin muscles at the lateral epicondyle, combined with the absence of shoulder pain, were the most discriminating factors for lateral epicondylitis (Helliwell, Bennett, Littlejohn, Muirden, & Wigley, 2003). A number of clinical provocative tests for lateral

epicondylitis have been suggested in the literature, and have been summarized in Table 1.

The most universal provocative test for lateral epicondylitis is pain reproduced with resisted wrist extension with the forearm in pronation; which is termed Cowen's test (Geoffroy, Yaffe, & Rohan, 1994) (Figure 1). There is often significantly more pain reproduced if the elbow is in an extended position, rather than a flexed position (Boyer & Hastings, 1999; Burgess, 1990; Coonrad, 1986; Nirschl, 1973; Stratford, Levy, & Gowland, 1993). Almost universally, there is accompanying localized tenderness to palpate near the origin of the extensor carpi radialis brevis and extensor carpi ulnaris tendons just anterior and distal to the lateral epicondyle (Boyer & Hastings, 1999; Burgess, 1990).

Figure 1. Cowen's Test, from www.ucbones.com

Other recommended clinical tests for lateral epicondylitis include: Mill's test, in which lateral elbow pain reproduced by stretching of the common extensor origin by the combined movement of extending the elbow while flexing the wrist (Wadsworth,

1987) and pain reproduced by resisted extension of the middle finger (Haker & Lundeberg, 1991; Wadsworth, 1987). Gardner (1970) suggested a simple, but un-validated, "chair test" in which the patient is asked to lift a chair with their forearm in a position of forearm pronation and palmer wrist flexion. A sharp pain over the lateral aspect of the forearm was thought to be pathognomic of lateral epicondylitis. All these tests involve gripping, and grip strength has been shown to be a reliable measure of lateral epicondylitis severity (Pienimaki et al., 2002; Smidt, van der Windt, Assendelft, Mourits, et al., 2002).

No diagnostic imaging is usually required to confirm the diagnosis of lateral epicondylitis, however, if there is a history of significant trauma a plain radiograph is sometimes recommended to rule out the presence of pathology such as a fracture near the head of the radius, or osteoarthritis of the elbow. A systematic review by Pasternack, Tuovinen, Lohman, Vehmas, and Malmivaara (2001) concluded that there was limited evidence for the use of magnetic resonance imaging in diagnosing epicondylitis. This was mainly due to the small sample sizes of most studies and other methodological problems. Sonography is a more economical alternative for diagnosis of lesions in tendons such as the Achilles and infrapatellar tendons; however, clinical diagnosis based physical examination was still considered the 'gold standard' by Miller, Shapiro, Schultz, and Kalish (2002) for confirming the presence of lateral epicondylitis.

Table 1.

Clinical Tests for Lateral Epicondylitis

Clinical Tests/ Findings	**Description**	**References**
Chair test	Pain when patient asked to lift chair with forearm in a pronated postion.	(Gardner 1970)
Cowen's test	Pain with resisted wrist extension.	(Boyer & Hastings, 1999; Burgess, 1990; Coonrad, 1986; Nirschl, 1973)
Functional tests	Pain and weakness with resisted grasp.	(Pienimaki et al., 2002; Stratford, Levy, & Gauldie, 1987; Thurtle, Tyler, & Cawley, 1984)
Mill's test	Pain with stretching of the common extensor origin.	(Wadsworth 1987)
Resisted finger extension	Pain with resisted extension of the middle digit.	(Haker and Lundeberg 1991)
Tenderness with palpation	Tender to moderate pressure near or just distal to the common extensor origin.	(Boyer & Hastings, 1999; Burgess, 1990)

On review of the relevant research, many of the clinical provocative tests for lateral epicondylitis have not been definitively validated or their reliability tested. It was concluded, that for the purpose of this research study, the physical diagnosis of lateral epicondylitis be based on using a number of well recognized tests rather than on any single test. It also was important that any other potential causes for the patient's lateral elbow pain be excluded. Some possible clinical tests have been excluded, such

as pain with extension of the middle finger and Mill's test, as there were conflicting reports in the literature; with some authors feeling these tests are a positive indicator for lateral epicondylitis (Haker & Lundeberg, 1991; Wadsworth, 1987), while other authors suggesting these are a positive test for radial tunnel syndrome (Boyer & Hastings, 1999). Pain with an extended grasp was included as it is a very functionally relevant test for lateral epicondylitis, and grip strength has been commonly used as an outcome measure for treatment of lateral epicondylitis (Pienimaki et al., 2002; Stratford et al., 1987; Thurtle et al., 1984). Grip strength measurements also have demonstrated high generalizability coefficients for both interrepetition and interoccasion measurements in patients with lateral epicondylitis (Stratford, Norman, & McIntosh, 1989). Although simple tenderness to palpation over the anatomical origin of the muscles of the common extensor origin muscles is a subjective finding, this has consistently been reported as a physical finding confirming the presence of a lateral epicondylitis (Boyer & Hastings, 1999; Burgess, 1990). However, no tenderness should be noted over the deep interosseus nerve distal to the level of the radial head, as this may be an indicator of radial tunnel syndrome (Boyer & Hastings, 1999).

Differential Diagnosis

Lateral epicondylitis is the most common cause of lateral elbow pain; however, there are a number of other conditions which can mimic symptoms of lateral epicondylitis, and needed to be excluded in this study. One of the primary conditions to be differentiated from lateral epicondylitis is impingement of the deep branch of the radial nerve (posterior interosseous nerve) as it enters the supinator muscle in the

proximal forearm, termed radial tunnel syndrome. Radial tunnel syndrome can be distinguished from lateral epicondylitis by the location of maximal tenderness, which is below the level of the radial head rather than the lateral epicondyle, and by pain elicited over the area of the radial tunnel during resisted extension of the middle finger (Boyer & Hastings, 1999). Electromyography and nerve conduction studies have not been found to be helpful in differentiating radial tunnel syndrome, as these tests have poor specificity for this condition (Lister, Belsole, & Kleinert, 1979). Degenerative arthritis of the elbow joint is another possible cause of elbow pain and usually present as a diffuse aching, and often accompanied by a loss of elbow range of motion; rather than lateral elbow pain reproduced on resisted movements, as with lateral epicondylitis (Burgess, 1990). Considering the age group normally affected by lateral epicondylitis, it was also important to rule out pain referred to the elbow region due to degenerative cervical radiculopathy by clinical examination of the cervical spine, including neurological testing such as dermatome and myotome testing, and tendon reflexes (Cyriax, 1978). Referred pain from the cervical spine was suspected if there was a painful restriction of neck range of motion or neurological findings, along with negative findings that would normally be associated with lateral epicondylitis (Coonrad, 1986). Systemic diseases are a rare cause of tendon disorders, and metabolic and inherited diseases are reported to represent less than one percent of all chronic tendon disorders (Jarvinen et al, 1997). A sprain of the lateral collateral ligament of the elbow is another potential source of lateral elbow pain and was ruled out by a pain free varus stress test to the elbow. Posterolateral rotary instability of the elbow is also a rare but possible cause of lateral elbow pain when there is a history of trauma, and patients describe a vague feeling of instability. Testing for posterolateral

rotary instability was performed clinically by applying a valgus stress when the elbow is extended and forearm supinated which induces a rotary subluxation to occur. A palpable 'clunk' on elbow flexion as the instability reduces was considered a positive test (Boyer & Hastings, 1999).

Current Treatment Approaches

A confusing array of surgical and conservative treatments have been suggested for lateral epicondylitis. Over forty different treatment techniques have been reported in the literature (Sevier & Wilson, 1999). Initial treatment advice for lateral epicondylitis usually involves rest, ice (Rivenburgh, 1992), use of epicondyle straps and braces (Struijs et al., 2002), and anti-inflammatory medication (Green et al., 2002b). More resistant cases may receive anti-inflammatory injections with drugs such as cortisone (Assendelft et al., 1996; Smidt, Assendelft et al., 2002). Rehabilitation may include the use of modalities such as ultrasound (van der Windt et al., 1999), laser (Basford, Sheffield, & Cieslak, 2000; Hart & Hoens, 2002), phonophoresis or iontophoresis (Nirschl, Rodin, Ochiai, & Maartmann-Moe, 2003), magnetic field therapy (Devereaux, Hazleman, & Thomas, 1985), or acupuncture (Green et al., 2002a). Physical treatments for lateral epicondylitis have included friction massage (De Bruijn, 1984), stretching and eccentric strengthening exercises (Svernlov & Adolfsson, 2001). Manipulation of the elbow (Vicenzino, Paungmali, Buratowski, & Wright, 2001), or wrist (Struijs et al., 2003) or neck (Vicenzino, Collins, & Wright, 1996) also have been recommended in the literature.

Only approximately five percent of the most resistant cases may go for surgery such as a lateral release (Buchbinder, Green, Bell et al., 2002; Owens, Murphy, & Kuklo, 2001) or more recently, extracorporal shock therapy (Buchbinder, Green, White et al., 2002; Haake, Hunerkopf, Gerdesmeyer, & Konig, 2002; Speed et al., 2002). Nirschl (1992) suggested the following indications for surgery for the most resistant cases of lateral epicondylitis: exercise-induced pain for over a year, exercise-induced pain after more than three cortisone injections, failure to respond to an adequate course of conservative treatment, and an unacceptable quality of life or significant work disability. Patients who require multiple cortisone injections appear approximately twice as likely to go on to having surgery (Bowen, Dorey, & Shapiro, 2001).

It is difficult to determine if most treatments for lateral epicondylitis significantly alter the natural course of the condition (Bowen et al., 2001; Labelle et al., 1992). In a recent systematic review, it was concluded that there was only weak evidence for the efficacy of ultrasound in the treatment of lateral epicondylitis (Smidt et al., 2003). There was insufficient evidence, due to contradictory results, low subject numbers, and insufficient power, to demonstrate the benefit or lack of effect of treatment for most of the other studies examined.

Fine Motor Control

Fine motor control of the hand is provided by a complex and delicate, but amazingly adaptable, sensorimotor system (Lemon, 1993; Shumway-Cooke & Woolacott, 2000). The development of fine motor control in primates has a protracted maturation process. Although infants can perform gross reflexive grasping motions, the development of fine motor control of the hand does not develop for many months. It

is hypothesized that the acquisition of new motor skills parallels the development of the corticospinal system, and is reinforced through practice and motor learning (Donoghue, Hess, & Sanes, 1996). Grip formation in infants develops at approximately 10 to 22 weeks (Koslowski & Bruner, 1972), however, pincer grip does not develop until 9 to 13 months, and complex hand function develops over several years (von Hofsten & Fazel-Zandy, 1984)

The hand, with its enormous sensitivity, provides continuous sensory feedback to the central nervous system which in turn, helps modulate the motor system (Johansson & Westling, 1987; Westling & Johansson, 1987). Mulder's model (1991) of dynamic motor programming defines the sensorimotor system as a functional flexible and highly integrated system, in which sensory input from the environment and cognitive processes continuously interact with motor processes. The presence of pain can disturb this sensory-motor system resulting in increased compensatory movement patterns, a decrease in coordination, and difficulty with synergistic use of muscles (Graven-Nielsen, Svensson & Arendt-Nielsen, 2000; Smeulders, Kreulen & Bos, 2001; Sterling, Jull, & Wright, 2001).

The term fine motor control refers to the ability to do skilled movements with the hands and is related to the concept of manual dexterity (Trombley & Scott, 1989). Manual dexterity can be classified as fine dexterity, which is the ability to manipulate objects with the distal part of the fingers, and gross dexterity involving use of the more proximal joints (Backman, Mackie, & Harris, 1991; Desrosiers, Rochette, Hebert, & Bravo, 1997). Fine dexterity requires precise independent use of individual

digits, and requires precise spatiotemporal patterning of muscle activity (Bennett & Lemon, 1996; Gerloff, Corwell, Chen, Hallett, & Cohen, 1998). Based on the literature review, fine motor control as demonstrated by fine dexterity was the skill of interest in this study, as it was anticipated to be most affected by sensorimotor deficits.

Use-Dependent Neuroplasticty

The adult neurological system was traditionally viewed to be essentially static and unchangeable once development was complete; however, it is now appreciated that throughout life the nervous system is highly modifiable with use and experience, termed neuroplasticity. It is also recognized that neuroplastic changes occur at multiple levels in the peripheral and central nervous system, including at the spinal cord, brainstem, and cortical levels (Molinari, Filippini, & Leggio, 2002; Wolpaw & Tennissen, 2001).

Neuroplastic changes provide the mechanism for motor learning and normal development, and there is continual ongoing reorganization within the neurological system dependent on the physical environment and functional needs of the individual Riolet-Pedotti, Friedman, & Donoghue, 2000; Shadmehr & Holcomb, 1997). Several primate studies have shown that the normal somatotopic representation of body areas in the somatosensory cortex and motor cortex are modifiable with use (Nudo, Milliken, Jenkins, Merzenich, Ochs, Allard, & Guic-Robles, 1990; Jenkins, & Merzenich, 1996). Human studies also have also demonstrated dynamic nature of cortical representations with use (Classen, Liepert, Hallett, & Cohen, 1999;

Donoghue, 1995; Kaas, 1991; Mogilner et al., 1993; Sanes & Donoghue, 2000). In a study of the primary somatosensory cortex of Braille readers, representation of the right index finger of the Braille reading hand was larger when compared to their left hand, or to the right hand of control subjects using magnetoencephalography (Pascual-Leone & Torres, 1993). Further studies in this area found that representation of first dorsal interossei muscle of the reading hand of Braille readers in the primary motor cortex was enlarged compared to the other digits of the non-reading hand, or control subjects (Pascual-Leone et al., 1993). There also is significant functional reorganization of the somatosensory cortical representation following injuries such as a peripheral nerve or spinal cord injury (Chen, Cohen, & Hallett, 2002; Kaas, 2000), stroke (Nudo & Friel, 1999; Ploughman, 2002) or amputation (Schwenkreis et al., 2003). The primary mechanism for organizational changes within the cortex is thought to be a functional increase or decrease in synaptic strength, through the long-term potentiation (LTP) or long term depression (LTD) of synaptic activity (Abarbanel, Huerta, & Rabinovich, 2002; Buonomano & Merzenich, 1998; Hess & Donoghue, 1994). Long-term structural changes of the architecture of the cortex, due to biochemical and genetic factors, also are important in the long-term modification of synaptic connections (Chain et al., 1999; Kandel, Schwartz, & Jessel, 2000).

Neuroplastic Changes with Repetition

Neuroplastic changes are important for positively adapting to the environment and learning new behaviours (Daoudal & Debanne, 2003; Xerri, Merzenich, Jenkins, & Santucci, 1999). For example, subjects taught to practice a novel five-finger exercise on the piano, showed, over the course of five days, improvements in skill as

demonstrated by a decrease in the number of errors and an increased number of key strokes. These gains were accompanied by a significant increase of the cortical representation for the finger flexor and extensor muscles as mapped using transcranial magnetic stimulation (Pascual-Leone et al., 1995).

Although neuroplastic changes are generally beneficial, there is some research that has identified neuroplastic degradation of the normal orderly topology of the somatosensory cortex as a result of intensive, highly repetitive, stereotypical movements (Byl et al., 1996; Byl et al., 2000; Elbert et al., 1998; Jenkins et al., 1990; Pantev et al., 2001). Byl presented a model relating neuroplastic changes and tendonitis in her 1996 study with adult owl monkeys. In this study, two female owl monkeys were behaviourally trained to perform a repetitive reaching and grasping task to receive food pellets. Both monkeys performed between 1,100 to 3,000 attended, stereotypical opening and closing movements during a daily one to two hour session. Training continued several weeks until the accuracy of movement and specific task deteriorated to a success rate of less than 50 percent. Subjective signs of tremor, pain and difficulty opening and closing the hand were observed at this point. The monkeys were then anesthetized, and the cortical representation of the S1 cortical area 3b was mapped using an open craniotomy and detailed electrophysiological mapping techniques were then compared to cortical maps of normal control monkeys. The cortical representations of the 3b area were reported to be significantly enlarged and dedifferentiated in both these monkeys compared to the controls. The degraded cortex showed changes in the size and distribution of the receptive fields of individual digits, and a "blurring" of the normally segregated boundaries between

representations of individual digits. The authors attributed the difficulty in motor performance to these neuroplastic alterations in the somatosensory cortex. The monkey who developed the greatest representational changes in the 3b area, however, was reported to also develop signs of an apparent tendonitis. Theses signs were marked by behavioural changes such as the monkey only performing a limited number of trials in succession, and excessive attention to the hand with licking or sucking the thumb. The specific conditions described by the authors that were required for cortical neuroplastic changes included: cognitively attended, highly repetitive and stereotypical movement patterns, involving synchronous sensory inputs (N. Byl, Merzenich, & Jenkins, 1996; Byl & Melnick, 1997; Byl et al., 1997). Many manual-type occupations also require focused and repetitive hand-intensive movements, and appear to offer similar conditions as those required for neuroplastic remodelling in the adult monkey studies. Byl has speculated that dysfunctional repetition-induced cortical neuroplastic changes similar to those observed in experimental animal models may be involved in the pathogenesis of repetitive strain injuries in humans (Byl et al., 1997; Elbert et al., 1998). Perhaps a decline in motor performance worsens chronic tendon disorders by increasing the duration of loading of an already compromised tendon unit (Barbe & Clark, 2004; Ardnt-Nielsen, Duarte, & Graven-Nielsen, 2004).

There are limited studies investigating effects of highly repetitive movement patterns on cortical reorganization in humans. The effect of repetition has, however, been investigated in musicians due to the intensity and cognitively focused manner in which they practice (Munte, Altenmuller, & Jancke, 2002; Schlaug, 2001). Intensive

musical training in professional musicians has been associated with an enlarged representation in the somatosensory cortex for the fingers used to play their particular instrument (Pantev, Englelein, Candia & Elbert, 2001; Pujol et al, 2000). Some musicians, however, develop fine motor control problems, termed occupational hand cramps or focal hand dystonia, which may be associated with repetition-induced neuroplastic changes involving dedifferentiation of the representational map of the somatosensory cortex (Elbert, 1998; Byl, Nagaragan, 2000; Byl, 2004). Unlike tendinitis, however, focal hand dystonia is a non-painful condition and not usually associated with peripheral tissue injury (Barr & Clark, 2004). Focal hand dystonia may also arise from several possible mechanisms other than repetitive use, such as genetics, environmental factors, or disorders of the basal ganglia; and the exact relationship between focal hand dystonia and repetitive use remains unclear (Beradelli et al., 1998; Waddy et al., 1991).

Barbe et al. (2003) demonstrated, using a rat model, that pathological changes in muscles were accompanied by a concomitant loss in motor performance. In this study, rats were trained to repeatedly reach to retrieve their food through a tube. The rate of retrieval was recorded, and the techniques that the rats used were monitored. Upper extremity tissue sample were taken at specific intervals for morphological and histological examination. There was significant decrease in reach rate by the end of week five, and the rats were observed to have begun using a gross raking pattern to retrieve the food pellet, rather than their previous scooping strategy with the fine muscles of the hand. Concurrently, morphological changes in the muscles of the reaching forelimb including a fraying of myofibrils were noted, and histological

evidence of an inflammatory response was measured (Barbe et al., 2003). The authors commented that this decrease in fine motor control may have been the result of peripheral tissue injury, or cortical dedifferentiation as elucidated by Byl et al (1996).

Motor Control and Tendonitis

Individuals experiencing musculoskeletal pain can exhibit alterations in motor responses. This may involve changes in coordination, a decrease in movement amplitude and velocity due to pain, abnormal recruitment of the antagonist and agonist muscles, and difficulty with synergistic muscle use (Graven-Nielsen, Svensson, & Arendt-Nielsen, 2000; Smeulders, Kreulen, & Bos, 2000; Sterling & Jull, 2001). These motor changes due to pain are related to the "pain-adaptation" model described by Lund, Donga, Widmer & Stohler (1991). However, the exact mechanisms by which pain affects motor control, still appears under investigation. Motor control has been researched in some chronic soft tissue injuries. For example, differential motor control of the superficial and deep muscles of the abdominal and lumbopelvic regions has been consistently found in subjects with low back pain (Ferreira, P.H., Ferreira, M.L., & Hodges, 2004; Hodges & Moseley, 2003). In a human experimental model, pain induced by injection of hypertonic saline into the m. biceps brachii and m. triceps brachii resulted in perturbations of the motor control with abnormal activation of agonist and antagonist muscles and difficulties in motor control (Ervilha, U., Arendt-Nielsen, L., Duarte, M.& Graven-Nielsen, L., 2004). Motor control impairment has also been observed, and measured in patients with chronic undifferentiated wrist pain (Smeulders, Kreulen, & Bos, 2001; Smeulders, Kruelen, Hage, Rutt, & Mulder, 2002).

Very few researchers have measured motor control in patients with tendonitis. Viikari-Juntari et al. (1994) compared the manual dexterity in 26 meat cutters and packers with a history of two or more episodes of wrist tenosynovitis to a control group matched for gender, occupation, age, and job seniority, and found no significant differences. The measures used in this study included measures of reaction time, movement time, visual attention and visuospatial ability. Subjects in the experimental group had a history of tenosynovitis but were asymptomatic at the time of testing. The results suggest that poor manual dexterity may not be a predictor of wrist tenosynovitis. Tenosynovitis also is an inflammatory process involving only the sheath surrounding the tendon and does not involve damage to the tendon structure, thus it may not be representative of chronic tendon injuries such as lateral epicondylitis.

Pienimaki, Kauranen, and Vanharanta (1997) measured the motor performance of patients with chronic unilateral lateral epicondylitis, and concluded that reaction speed and speed of movement were decreased bilaterally in patients when compared to age and gender-matched control subjects The authors were unable to explain why there was a bilateral decrease in patients with unilateral symptoms, but they suggested that an overall decrease in motor performance may have been a predisposing factor for the patient developing lateral epicondylitis. However, the authors did not control for effect of hand dominance, and the measures used were tests of general gross motor control and likely not sensitive to deficits in fine motor control.

Summary of Literature Review

Lateral epicondylitis is a frequently occurring overuse injury associated with repetitive hand use, and is a common cause of work-related disability (Chiang et al., 1993; Dimberg et al., 1989; Gilbert, Tick, & VanEerd, 1997; Kivi, 1984). The functional anatomy and clinical diagnosis of lateral epicondylitis are well-documented in the literature; however, there appears to be a limited understanding of the pathological processes. The natural history of lateral epicondylitis is variable, with some subjects responding to mechanically based interventions such as exercise or braces, while others fail to respond to treatment. The absence of a universally recognized and successful treatment approach for lateral epicondylitis, and the failure of some clients to respond to multiple forms of therapy, suggests there may be other factors (besides mechanical) involved in resistant non-resolving cases. Motor control problems have been implicated in other soft tissue injuries (Ervilha, Arendt-Nielsen, Duarte, & Graven-Nielsen, 2004; Ferreira, P.H., Ferreira, M.L., & Hodges, 2004; Hodges, Moseley & Lorimer, 2003; Smeulders, Kreulen, & Bos, 2001; Smeulders, Kruelen, Hage, Rutt, & Mulder, 2002) and the complaints of many patients of a feeling of clumsiness, suggests that perhaps some clients with lateral epicondylitis may experience difficulty with fine motor control. It appears to this author that the relationship between fine motor ability and tendonitis in humans is unclear and largely unstudied. Research is needed to systematically investigate if there are objective differences in fine motor control between individuals with tendonitis, in this case lateral epicondylitis, and those individuals without tendonitis, using valid and reliable measures.

PURPOSE

The purpose of this study was to examine whether there were differences in fine motor control ability between individuals with lateral epicondylitis and control subjects matched on age, gender and hand dominance, using objective, validated measures.

HYPOTHESIS

The following hypothesis was tested:

> There would be significantly less fine motor control ability in the group with lateral epicondylitis compared to the matched control group; as measured by the Purdue Pegboard and the Complete Manual Dexterity Test.

Delimitations

This study was delimited to subjects between the ages of 30-53 years who experience an occupationally-induced lateral epicondylitis in their upper extremity, and were referred for physical therapy treatment in one of two clinical locations in Alberta between June and December 2004. This study was also restricted to subjects who voluntarily agreed to participate.

Limitations

The results of this study do not apply to:

- subjects younger than 30, or older than 53, years of age
- those who have developed lateral epicondylitis from non-occupationally related causes
- people with lateral epicondylitis who experience a natural resolution of their symptoms
- people with lateral epicondylitis who have not been referred for physical therapy treatment
- all clients who are referred to physical therapy for treatment of lateral epicondylitis, since participation is non-random and voluntary

Factors Affecting Fine Motor Control

The measures used in this study, the Purdue Pegboard (PPT) and the Complete Manual Dexterity Test (CMDT) were initially developed to assess the suitability of applicants for industrial occupations. The normative data for the PPT and CMDT were first derived from the testing of specific occupations such as assembly jobs and production work (Tiffen, 1948), but were later applied to other situations. such interests as developmental disabilities in children (Gardner & Broman, 1979; Leslie, Davidson, & Batey, 1985) and for vocational rehabilitation purposes in adults (Hamm & Curtis, 1980). The available normative data, and other literature, were reviewed by the author to ascertain the effects of age, gender and hand dominance on fine motor control to determine whether these factors might have been confounding variables in this study.

Effect of Age on Fine Motor Control

Children and adolescents predictably show a greater variability in their fine finger dexterity related to age than adults, due to the protracted developmental process for fine motor control (Gardner & Broman, 1979). In a study by Hamm & Curtis (1980) of adult males and females referred for vocational rehabilitation, candidates under the age of 35 years appeared to have slightly better scores on the Purdue Pegboard Test than those candidates above the age of 35 years (Table 2). Tests of fine finger dexterity related to the PPT, such as the Grooved Pegboard Test and the Rate of Finger Tapping Test, have indicated that finger dexterity is relatively consistent in

both adult-aged men and women to about the age 55, after which time there is a significant drop for both genders (Kauranen & Vanharanta, 1996; Ruff & Parker, 1993) (Table 3).

Table 2.

Purdue Pegboard (Means and Standard Deviations) for Candidates for Vocational Rehabilitation by Age Category and Gender, number of pins placed in 15 seconds Adapted from Hamm & Curtis (1980)

	Candidates 35 Yr. and Younger		Candidates 35 Yr. and Older	
	Male, N = 116	**Female, N = 102**	**Male, N = 60**	**Female, N = 62**
Right	13.59 ± 1.25	15.28 ± 2.41	12.96 ± 1.81	14.08 ± 2.22
Left	13.18 ± 3.84	14.49 ± 2.13	11.90 ± 3.02	15.14 ± 4.19

Effect of Gender on Fine Motor Control

Some studies have shown a small effect of gender, although the results are sometimes contradictory. Women have been shown to score slightly better than men on tests of finger dexterity such as the Purdue Pegboard Test (Hamm & Curtis, 1980; Tiffen, 1948) and Finger Tapping Test (Ruff & Parker, 1993). Other studies also using the Rate of Finger Tapping Test, however, appear to illustrate that men have slightly better scores than women (Kauranen & Vanharanta, 1996) (Table 3). More recent research looking at the effect of age and gender using the Purdue Pegboard Test failed

to reveal any effect of age or gender on finger dexterity (Haward & Griffin, 2002). In general, age appears to be a greater predictor of performance on tests of fine motor control than gender, and scores on tests of fine motor control appears to increasingly decline for both genders over the age of 50 years (Kauranen & Vanharanta, 1996; Ruff & Parker, 1993).

Table 3.

Rates of Tapping per Second (Means and Standard Deviations) by Gender, Age and Limb, Adapted from Kauranen & Vanharanta (1996)

Limb	Gender	21 - 30 yr.	31 – 40 yr.	41 – 50 yr.	51 – 60 yr.	61 – 70 yr.
Left	**Male**	5.4 ± 0.5	5.7 ± 0.7	5.6 ± 0.7	4.9 ± 0.8	4.7 ± 0.9
	Female	5.1 ± 0.5	5.2 ± 0.4	5.3 ± 0.6	4.5 ± 0.7	4.6 ± 0.7
Right	**Male**	5.8 ± 0.6	6.0 ± 0.6	6.2 ± 0.6	5.4 ± 0.9	5.2 ± 0.8
	Female	5.7 ± 0.6	5.6 ± 0.5	5.7 ± 0.6	5.1 ± 0.8	5.0 ± 0.7

Note: N = 200, with 20 males and 20 subjects in each age category

Effect of Hand Dominance on Fine motor Control

Handedness is usually considered an expression of cerebral lateralization, and human studies using magnetoencepholgraphy have shown a larger representation of the hand area in the primary motor cortex of dominant hemisphere than the contralateral hemisphere controlling the non-dominant hand (Reiss & Reiss, 2000; Volkmann,

Schnitzler, Witte, & Freund, 1998). Conventional thought would suggest that a person's ability on tests of fine motor control would be better in the dominant limb due to a practice effect; however, on examination of the literature, the effect of hand dominance on fine motor control is not completely understood.

In one study, investigating the relationship between the scores of subjects on the Purdue Pegboard Test and hand preference scores, reported a strong correlation, suggesting that hand preference and motor performance are related (Triggs, Calvanio, Levine, Heaton, & Heilman, 2000). In another study using the PPT , investigating the fine motor performance in 22 right-handed and 22 left handed persons, the results indicated that for right-handed individuals the performance of the dominant hand (mean 14.09, SD = 1.72) was significantly better than the non-dominant (mean 13.09, SD = 1.87). In left-handed individuals there were similar but smaller performance differences between the dominant (mean 13.82, SD = 2.15) and non-dominant hands (mean 13.59, SD = 1.68) (Judge & Stirling, 2003). In other words, the effect of dominance on motor performance appeared stronger in right-handed subjects than left-handed subjects. Chan (2000) used the Purdue Pegboard to investigate the finger and manual dexterity of the right and left hands of 30 male and 30 female college students, in which 10 percent of the subjects were left-hand dominant subjects. This study reported a statistically significant difference between the mean scores for the right hand (mean 19.6, SD = 6.19) compared to the left hand (mean 16.8, SD = 4.50) in females ($p < .01$), but not in males; right hand (mean 16.8, SD 2.49), left hand (mean 16.1, SD = 2.12). Ruff & Parker in 1993, studied the fine motor control ability of 360 normal volunteers using a similar test to the PPT, called the Grooved Pegboard

Test, and failed to demonstrate an effect for hand dominance. A four-way analysis of variance for the independent variables of age, educational level, gender and hand dominance, did not show a main effect for hand dominance or an interaction effect with the other variables (Ruff & Parker, 1993). In a study by Shahar (1998) comparing the PPT scores of 54 adults with traumatic hand injuries to healthy control subjects, no significant difference was found between the mean scores for the dominant-injured and the non-dominant-injured groups ($t = 0.822$; $p = 0.41$) (Table 4). Overall, it appears that there may be several possible factors influencing the effect of hand dominance on fine motor control, including such factors such as gender and handedness.

Table 4.

Purdue Pegboard Test (Mean Scores and SD) for Dominant-injured (n = 32), and Non-dominant-injured (n = 22) subjects, Adapted from Shahar, Kizony & Nota (1998)

	Dominant Injured		**Non-dominant Injured**	
	Mean	**SD**	**Mean**	**SD**
Dominant hand	10.63	3.30	14.36	2.65
Non-dominant hand	13.25	1.88	13.51	1.86

Summary of Literature on Age, Gender and Dominance

The normative data comparing age and fine motor control ability illustrates a relatively stable performance on tests of fine motor control between the ages of 30 and 55, with a natural break point with those individuals 55 years of age and older having a more significant decline in finger dexterity. The age range for inclusion in this study was therefore chosen to be between the ages of 30 and 53 to avoid the significant decline at the age of 55 years. The age range of this study also incorporated the age range of the individuals most likely to suffer from lateral epicondylitis (Assendelft et al., 1996; Gilbert et al., 1997; Higgs & Mackinnon, 1995; Viikari-Juntura, 1984). The results of the research on the effect of gender on finger dexterity appears inconclusive, and it appeared reasonable that the effect of gender be controlled for in this study as this factor may be a confounding variable. (Hamm & Curtis, 1980; Haward & Griffin, 2002; Stewart et al., 1999; Tiffen, 1948). The literature on the effect of hand dominance on fine motor control ability also appears unclear. On some tests of finger dexterity, motor performance was better when using the preferred hand of the individual (Triggs, Calvanio, Levine, Heaton, & Heilman, 2000), however, gender may also be a factor influencing the overall effect of hand dominance (Chan 2000). Whether a person is left-hand or right-hand dominant may also influence the degree to which hand dominance affects fine dexterity, possible due to hemisphere asymmetries of motor control. Finally, other researchers such as Ruff & Parker (1993) and Shahar, Kizony & Nota (1998) found no effect for hand dominance in their studies. Nevertheless, it was considered important to control for the potential effect of hand dominance in this research.

In conclusion, the available research literature and normative data suggested that age, gender, and hand dominance were probable confounding variables which could influence the test results and subsequent interpretation. Thus, age, gender and hand dominance were controlled for in this study.

ETHICAL CONSIDERATIONS

The potential physical or psychological risk to the research subject was minimal. There was a potential risk for subjects in the tendonitis group involving a possible temporary exacerbation of their symptoms, similar to what may occur during physical therapy treatment. Participation in the study was entirely voluntary, and a subject could withdraw at anytime without affecting the treatment of their lateral epicondylitis or informing their treating therapist. The intent and purpose of the study was thoroughly explained to each participant, and a description of the results of the study will be supplied on request.

Confidentiality of records and scores was strictly maintained. Records are stored in a locked secure file cabinet at Okanagan University College, and will be stored in a secure location in the Department of Physical Therapy, University of Alberta following completion of the study. Each subject was assigned a unique case number to ensure confidentiality. This study was reviewed prior to implementation by the Human Research Ethics Committee of the University of Alberta and the Research Ethics Board of the Okanagan University College, and their recommendations and guidelines were followed.

METHODS

Design

The research design of this study is quasi-experimental, in that unlike a true experimental design, the independent variable (i.e. presence or absence of lateral epicondylitis) is not truly manipulated. The experience of many practicing clinicians who regularly use the Purdue Pegboard Test is that the original norms that are supplied with the test are often higher than those test scores observed in clinical practice. The grouping of subjects in the normative data supplied by the manufacturer includes subjects of widely different characteristics, such as college students and veterans. It has been recommended by (Hamm and Curtis (1980) that a clinical sample be compared to its own normative population. Z – tests performed by these authors between the scores for the candidates for vocational rehabilitation and the normative data provided for the PPT were significant ($p \leq .05$), supporting the importance of comparing the PPT scores of a clinical sample with their own normative sample.

For these reasons, a control group matched for age, gender and hand dominance was used in this study. The normative data supplied with the Purdue Pegboard Test separates scores for the dominant and non-dominant hand for children and adolescents up to the age of 16 years, however, does not distinguish between the dominant and non-dominant hands in the information provided for adult-aged workers. The normative data supplied with the CMDT also does not offer normative data for the dominant and non-dominant hands. This provided an additional reason to

use a hand-dominance-matched control group in this study. Hand dominance was determined by the most common method - which is the hand the person uses for writing (Reiss & Reiss, 2000).

Sampling

The sampling technique for the experimental group involved non-random selection of patients referred for treatment of lateral epicondylitis to Cumulative Activity Related Disorders (CARD) programs at two locations; Millard Health in Edmonton, and Orion Health in Calgary. Subjects in the control group were non-randomly recruited from patients attending local physiotherapy clinics for non-upper extremity related conditions: Sun City Physiotherapy and Columbia Health in Kelowna. Test data from the lateral epicondylitis group were first collected; followed by collection of data from the control group.

Sample Size

Effect sizes based on review of five relevant previous studies appeared large (Desrosiers, Hebert, Bravo, & Dutil, 1995; Hamm & Curtis, 1980; Kauranen & Vanharanta, 1996; Pienimaki et al., 1997; Ruff & Parker, 1993). Table 2.3.2 for t-tests from Cohen's book on statistical power analysis was used to determine sample size (Cohen, 1988). Based on an alpha value of 0.05, and an estimated effect size of .80, a minimum sample size of 20 subjects in the experimental group and control group was estimated to achieve a power of .80 (Appendix A). A larger sample was collected to ensure that there were at least 25 subjects in each group, anticipating that some of the data might be unusable.

Subjects

All subjects were recruited voluntarily by way of a poster inviting the patient to participate if they wish. If interested, the potential participant was given further information from the receptionist regarding the study. The subjects in the experimental groups were adult-aged injured workers with lateral epicondylitis who were attending a Cumulative Activity-Related Disorder (CARD) program. The control subjects were individuals attending physiotherapy treatment for a non-upper extremity condition, and were selected to provide an equal representation of ages and gender as in the experimental group.

Twenty-eight subjects with lateral epicondylitis, who met the following inclusion and exclusion criteria, were included in this study. The mean age of subjects in the lateral epicondylitis group was 41.93 years (SD = 6.4 yrs) and ranged between 30 and 53 years. Twenty-eight matched control subjects, who matched the inclusion and exclusion critieria, were included in this study, with mean age of 42.36 years (SD = 6.44 yrs) and ranged between the ages of 30 and 52 years. The gender distribution was 57% female and 43% male in both the LE and control groups. The mean time since injury for subjects in the LE group was 30.54 weeks (SD = 36.69 weeks). The raw data for ages is provided in Appendix B, and further descriptive data is provided in Table 5.

Inclusion and Exclusion Criteria

Diagnostic Criteria:

For the purpose of this study, the following diagnostic criteria were needed to confirm the clinical diagnosis of lateral epicondylitis:

- Pain on the Cowen's test, as described – localized lateral elbow pain with resisted isometric wrist extension with the elbow extended (Boyer & Hastings, 1999; Burgess, 1990; Coonrad, 1986; Nirschl, 1973).
- Pain localized over the lateral epicondyle with maximal grasping with an extended elbow (Pienimaki et al., 2002).
- Anatomical tenderness to palpate the extensor carpi radialis brevis and extensor carpi ulnaris tendons near their origin (Boyer & Hastings, 1999; Burgess, 1990).
- Exclusion of cervical or shoulder disorders which may affect arm function by a clinical assessment by a trained physiotherapist (Cyriax, 1978; Helliwell et al., 2003).
- Exclusion by assessment of other sources of lateral elbow pain such as acute sprain of the lateral collateral ligament of the elbow, radial tunnel syndrome, or posterior rotary instability.
- Exclusion by history and clinical assessment of other conditions which may affect arm function such as a previous fracture of the arm, carpal tunnel syndrome, or systemic diseases.

Lateral Epicondylitis (Experimental) Group:

Include: an adult worker between 30 and 53 years of age and either gender with a confirmed clinical diagnosis of lateral epicondylitis; who are attending a treatment program for their condition, and are in good general health.

Exclude: those individuals with motor control problems due to: secondary medical conditions such as carpal tunnel syndrome or other peripheral nerve conditions, multiple sclerosis, Parkinson's disease, brain tumors, cerebral vascular accidents, peripheral neuropathies or other known neurological conditions. Also exclude those individuals on medications which may affect fine motor control such as tricyclic antidepressants, neuroleptic and antipsychotic drugs, and those withdrawing from use of alcohol or street drugs.

Control Group:

Include: an adult working population between 30 and 53 years of age and of either gender, who are attending physiotherapy treatment for a condition other than an upper extremity condition. No known medical or physical condition that might interfere with fine motor control, and in good general health

Exclude: those individuals with motor control problems due to secondary medical conditions such as carpal tunnel syndrome or other peripheral nerve condition, multiple sclerosis, Parkinson's disease, brain tumors, cerebral vascular accidents, peripheral neuropathies or other known neurological

conditions. Also exclude those individuals on medications which may affect fine motor control such as tricyclic antidepressants, neuroleptic and antipsychotic drugs, and those withdrawing from use of alcohol or street drugs.

PROCEDURES

Outcome Measures

Several potential measures of fine motor control were considered for this study, including: the O'Connor Finger Dexterity Test, the Grooved-Pegboard Test, the Valpar VCWS 204 Fine Finger Dexterity Test, the Purdue Pegboard Test (PPT), and the Complete Manual Dexterity Test (CMDT). The PPT and The CMDT were chosen, because they were thought to be the most sensitive tests for identifying difficulties in fine motor control of the digits. They are both well standardized tests, and practical for clinical use (Apfel & Carranza, 1992). The PPT was described by Fleishman and Ellison (1962) as the most sensitive test for measuring finger dexterity as it involves the "ability to make rapid, skilful, controlled manipulation of small objects, where the fingers are primarily involved" (p. 101). The recommendations of the American Society of Hand Therapist for the clinical assessment of fine motor control also supports the use of both the PPT and the CMDT as practical, reliable, and valid tests of hand function (Apfel & Carranza, 1992).

The Purdue Pegboard Test consists of a rectangular board with two vertical lines of pin holes. On the top of the board are four shallow wells containing pins, collars and washers. The PPT assesses a person's ability to use their fingers, wrists and arms in a

rapid repetitive manner, and includes three subtests: using either the right or left hand to place pins, placing pins using both hands simultaneously, and an assembly of pins, collars and washers using both hands to assist. The Complete Manual Dexterity Test consists of a folding board with 60 wells for medium sized cylindrical blocks to be placed into. The CMDT measures both gross and fine dexterity and consists of five subtests: the One-handed Turning and Placing test, the Turning test, the Displacing Test, and the Two-handed Turning and Placing test. Because this study compared the finger dexterity of the involved limb in an individual with lateral epicondylitis to the finger dexterity of a normal control subject, while controlling for the effect of hand dominance, the One-handed Pin Placement and the One-handed Turning and Placing tests were selected to avoid the bimanual use of the hands, and limit this possible confounding factor. Included in Appendix C is further information regarding the historical development and literature supporting the reliability and validity of the PPT and the CMDT.

In summary, the independent variable in this study is the presence or absence of lateral epicondylitis, and the two dependent variables are the measures used: the One-handed Pin Placement portion of the Purdue Pegboard Test, and the One-handed Turning and Placing portion of the Complete Manual Dexterity Test. The Purdue Pegboard which was used for testing during this study was Model 32020, manufactured by Lafayette Instrument (3700 Sagmore Parkway N., P.O. Box 5729 Lafayette, IN. 47903, www.lafayetteinstrument.com. The Complete Manual Dexterity Test which was used in Model 32023A, which is also manufactured by Lafeyette Instruments. The level of measurement for both measures is interval. Two separate

measures of fine motor control were chosen to increase the concurrent validity of this study. The Purdue Pegboard and CMDT were also chosen as they were inexpensive, easily administered, and clinically relevant.

DATA COLLECTION

Following informed consent, all testing was performed by the principal investigator. All subjects were screened using the previously described inclusion and exclusion criteria. The tests used for data collection included the One-handed pin placement portion of the Purdue Pegboard Test (PPT), and the One-handed Turning and Placing subtest of the Complete Manual Dexterity Test (CMDT). Both the Purdue Pegboard Test and the CMDT have detailed instructions as to how to setup and administer the test, which were followed by the investigator. The verbal instructions given to the test subjects are well documented in the instructions to ensure consistency between subjects. For the purpose of obtaining the greatest test reliability, three trials for the Purdue, and four trials of the CMDT, were administered in this study. The score for one trial of the PPT is the number of pins placed in thirty seconds. The score for one trial of the CMDT is the length of time in seconds necessary to turn and place 60 blocks. The subject completed three trials of the single handed pin placement portion of the Purdue Pegboard Test, and the cumulative score in seconds was obtained simply by adding of the three individual scores (Instructions and Normative Data for Model 32020 Purdue Pegboard. LaFayette, IN, Lafayette Instrument). Similarly, for the CMDT, the overall score on the test was the cumulative time in seconds required to complete all four administered trials (The CompleteManual Dexterity Test:

Examiner' Manual, 1969, Lafayette Instrument). The time required for the practice trial was not included in the scoring.

During the summer of 2004, 20 subjects with lateral epicondylitis were tested at Millard Health in Edmonton, and 18 subjects with LE were tested at Orion Health in Calgary. Data from ten (10) of the subjects in the lateral epicondylitis group were excluded for the following reasons:

- one (1) subject was excluded due to complaint of increased elbow discomfort during testing
- one (1) subject was excluded due to shoulder complaints in the affected limb
- one subject (1) was excluded due to symptoms of probable radial tunnel syndrome which arose during testing
- one subject (1) was excluded due to difficulty understanding instructions due to English as a Second Language (ESL).
- six (6) subjects were excluded as there was no age or gender match in the control group

During the fall of 2004, data from 23 control subjects was collected locally at Sun City Physiotherapy, and 8 subjects from Columbia Health. Data from three (3) subjects in the control group were excluded due to:

- two (2) subjects were excluded as there were no age or gender match in the LE group

- one (1) subject was excluded as symptoms of ulnar neuropathy arose during testing

Matching of Experimental and Control Groups

Both genders were included in this study, and the subjects in the experimental group were matched to a same gender subjects in the control group for comparison. Matching for age was achieved by individually selecting the control subject with the closest available match for age to the individual with lateral epicondylitis (Appendix B). The effect of hand dominance was controlled for by testing both the dominant and non-dominant limbs in the control subject, and then matching the affected limb of the experimental subject to the appropriate limb of the control subject. Matching of the experimental and control groups by right-handedness or left-handedness was not performed, as the intent was to control for the possible effect of hand dominance; not the effect of cerebral lateralization. In reality, only one subject in the lateral epicondylitis group was left hand dominant and the inclusion or exclusion of this subject would not have substantially altered the results of this study. Thus, twenty-eight (28) individuals in the lateral epicondylitis group were successfully matched to twenty-eight (28) control subjects on the variables of age, gender and hand dominance. This sample of 28 individuals was comprised of 12 males and 16 females. Appendix B illustrates the matched pairs.

ANALYSES

Statistical Analyses of the data was performed using the SPSS Ver 12.0 software (SPSS for Windows, SPSS, Chicago, IL). Standard descriptive statistical analyses were used to describe the characteristics of both groups, including: mean age, percentage male and female, and the mean time since injury for the lateral epicondylitis group. A two tailed t-test was used to determine if there was a significant difference between the mean ages of the lateral epicondylitis and control groups.

Inferential statistical procedures were used to test the hypothesis, and included a Two-way ANOVA to test for the main effect for the factors of hand dominance and group, and the interaction effect between group and affected limb, for each of the two measures (PPT and CMDT). A One-way ANOVA was performed on the experimental group to test whether there was an effect for the time since injury. The experimental group was divided into two sub-groups, those who had lateral epicondylitis for twelve weeks or less (acute group, n = 13and those individuals who had lateral epicondylitis for greater than twelve weeks (chronic group, n = 15) for both measures (PPT and CMDT).

RESULTS

There were no significant differences between the mean ages of the lateral epicondylitis group (41.98 ± 6.44 years) and the control group (42.36 ± 6.44 years) (t_2 = - 0.25, p = .80 (Table 4). The percentage of each gender was identical between the lateral epicondylitis group and the control group by design (Table 5).

Table 5

Descriptive Analysis of Sample Characteristics

Variable	Lateral Epicondylitis group n = 28	Control group n = 28
Age (SD)	41.93 years (± 6.44)	42.36 years (± 6.44)
Female, n= (% sample)	n = 16 (57%)	n = 16 (57%)
Dominant limb	(R) = 27	(R) = 27
	(L) = 1	(L) = 1
Affected limb	Dominant n = 19	N/A
	Non-dominant n = 9	
Weeks since injury (Range, SD)	30.54 ± 36.69 weeks	N/A

Table 6 below provides the mean scores and standard deviations for the Purdue Pegboard and CMDT test scores, for the dominant-affected, and non-dominant-affected limbs for the lateral epicondylitis group; and the corresponding data from the matched control group. For the reasons previously described, a comparison of the tests scores obtained in the control group to the available normative data was not meaningful, and there is a need for new normative studies. The data obtained in this

study indicate that subjects with lateral epicondylitis in their dominant limb placed on average 5.06 fewer pins over three trials of the PPT, and were on average 48.52 seconds slower than the control group to complete four trials of the CMDT, than individuals in the control group. Subjects with LE in their non-dominant limb placed an average of 4.89 fewer pins, and were 56.34 seconds slower than the control group.

Table 6

Mean Test Scores and Standard Deviations for Lateral Epicondylitis and Control Groups Dominant and Non-dominant Affected limbs

Affected Limb	**Lateral Epicondylitis Group**	**Control Group**
Dominant n = 19		
Purdue	42.05 (SD ± 7.08)	47.11 (SD ± 3.69)
CMDT	348.68 (SD ± 50.18)	300.16 (SD ± 43.3)
Non-Dominant n = 9		
Purdue	40.11 (SD ± 6.49)	45.00 (SD ± 3.28)
CMDT	367.78 (SD ± 38.55)	311.44 (SD ± 26.21)

The results and inferential analysis related to the hypothesis, as outlined on page 36, are provided below. Two-way ANOVAs were used for each measure (PPT and CMDT) to determine if there were significant differences in the outcome measures as a function of group and affected limb (see Tables 8 and 9 for ANOVA results). There

was no significant effect noted for whether the dominant or non-dominant limb was affected, with either the Purdue scores $F(1,52) = 1.66$, $p > 0.20$, or the CMDT scores $F(1,52) = 1.52$, $p > 0.22$. There also was no interaction effect between the group and the affected limb for the Purdue $F(1,52) = .003$, $p > 0.95$, or the CMDT $F(1,52) = 0.10$, $p > 0.75$. Therefore, as a result of the absence of the main effect for dominance, and the lack of an interaction effect, the scores on the PPT and CMDT for the dominant and non-dominant limbs were collapsed to represent only two groups: the lateral epicondylitis group (n = 28), and the control group (n =28). The combined scores (N, Mean, SD and Confidence Intervals) are provided below.

Table 7

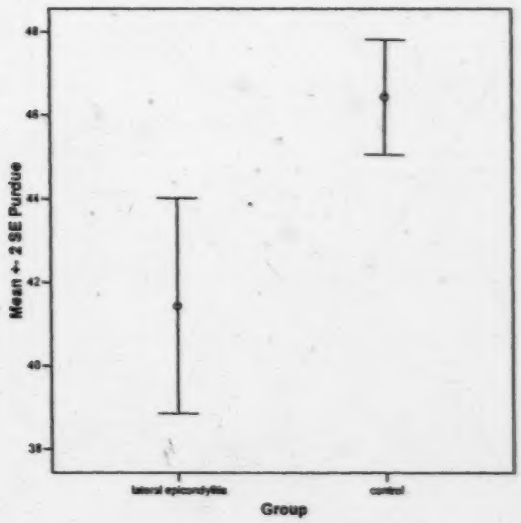
Mean Test Scores and Standard Deviations for Lateral Epicondylitis and Control Groups with Scores with Dominant and Non-dominant Affected Limbs Combined

		N	Mean	SD	95 % Confidence Interval for the Mean lower upper	
Purdue*	lateral epicondylitis	28	41.43	6.84	38.78	44.08
	control	28	46.43	3.65	45.01	47.84
CMDT**	lateral epicondylitis	28	354.82	46.91	336.63	373.01
	control	28	303.79	38.49	288.86	318.71

The results indicated a significant difference between the mean scores of the lateral epicondylitis group and the control group, for both the PPT $F(1,52) = 9.98$, $p < .005$, and the CMDT $F(1,52) = 18.11$, $p < .001$, (Figures 2 and 3, Tables 8 and 9 respectively). The lateral epicondylitis group demonstrated a significant decrease in fine motor control ability on both measures compared with the age- and gender-matched control group.

Figure 2.

Comparison of Mean Purdue Scores for the Lateral Epicondylitis Group and Control Group, ± 1 Standard Error



$p < .005$

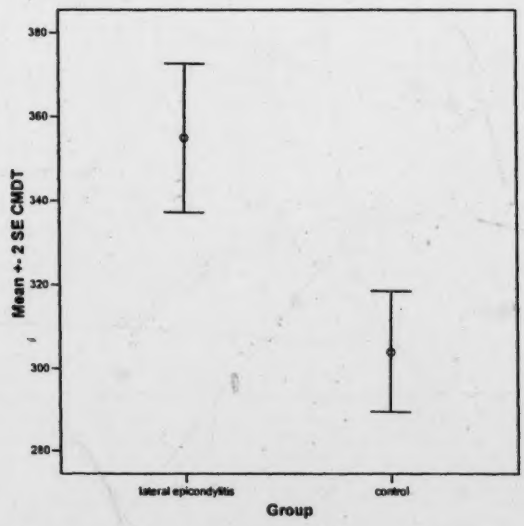
Table 8

Tests of Between - Subject Effects

Dependent Variable: Purdue

	df	Mean Square	F	p =
Group	1	301.796	9.985	.003**
Affected Limb	1	50.007	1.655	.204
Group x Affect Limb	1	.082	.003	.959

Figure 3 Comparison of Mean CMDT Scores for the Lateral Epicondylitis Group and Control Group, ± 1 Standard Error



$p < .001$

Table 9

Tests of Between - Subject Effects

Dependent Variable: CMDT

	df	Mean Square	F	p =
Group	1	33575.685	18.108	.000***
Affected Limb	1	2818.298	1.520	.223
Group * Affect Limb	1	186.114	.100	.753

Effect of Time Since Injury

The researcher questioned whether difficulties with fine motor control might be greater in those individuals who had lateral epicondylitis for a greater length of time (i.e. an effect for chronicity). The lateral epicondylitis group was, therefore, further divided into those subjects who reported they had their condition for less than or equal to 12 weeks (acute group n = 13), and those subjects with lateral epicondylitis for more than 12 weeks (chronic group n = 15). The group means and standard deviations are presented in Table 10 below. The scores for both measures were compared using a One-way ANOVA. There were, however, no significant differences between the acute lateral epicondylitis group and the chronic lateral epicondylitis group for both the PPT, $F(1,26) = .087$, and the CMDT, $F(1,26) = .094$, $p > .05$

Table 10.

Mean Test Scores and Standard Deviations for Acute and Chronic Lateral Epicondylitis Subgroups

		N	Mean	SD	95 % Confidence Interval for the Mean	
					lower	upper
Purdue[a]	acute	13	41.85	8.28	36.85	46.85
	chronic	15	41.07	5.59	37.97	44.16
CMDT[a]	acute	13	351.85	47.59	323.09	380.61
	chronic	15	357.40	47.84	330.91	383.89

[a] = indicates non-significant, $p > .05$

DISCUSSION

Repetitive strain injuries of the wrist and forearm are common work-related conditions that can be disabling and costly to treat (Barr & Barbe 2002). The commonly assumed model of injury for work-related lateral epicondylitis (LE) is a biomechanical overloading of the tendons of the common extensor origin, including the extensor carpi radialis brevis (ECRB), extensor carpi radialis longus (ECRL) and extensor digitorum communis (EDC) muscles. Repeated overuse, with inadequate time for the healing of microscopic partial tears, is thought to overwhelm the adaptive potential of the tendinous attachments of the muscles (Jarvinen et al., 1997; Melborn, 1998; Moore, 2002). The resistance of some cases of lateral epicondylitis to various

forms of conservative treatment may imply that this explanation is not true for all cases of LE. Complaints of clumsiness from some patients with lateral epicondylitis suggests that there may be alterations in motor control ability in these individuals (N. Byl et al., 1996; Byl & Melnick, 1997; Byl et al., 1997; N. N. Byl et al., 1996; Trembley, Mireault, LeTourneau, Pierrat, Bourrassa, 2002).

Few researchers have objectively tested and quantified fine motor control ability in individuals with tendonitis, specifically lateral epicondylitis, which was the purpose of this research. The mean age of the LE subjects (41.98 ± 6.44 yrs) and the propensity for the dominant limb to be most often affected (68 percent) was consistent with the findings of previous epidemiological studies, suggesting that the sample was likely representative of the larger population of individuals with lateral epicondylitis (Assendelft et al., 1996; Coonrad & Hooper, 1973; Rotoni, Fontana, Catamo, Noia, & Magnani, 2000). Subjects were compared to an age, gender and hand-dominance matched control group to ensure that these variables, which have been suggested to affect fine motor control, would be evenly distributed in both groups.

The results of this study demonstrated a significant decrease in fine motor control in subjects with lateral epicondylitis compared to the control group. Subjects with lateral epicondylitis placed approximately 10.5 percent fewer pins on the PPT, and demonstrated a 16.8 percent increase in time to complete the CMDT, compared to the matched control group. Results from both the PPT and the CMDT, two separate measures of fine motor control, showed similar decreases in fine motor control of

subjects with lateral epicondylitis compared to the control group. The fact that both measures showed similar findings increases the concurrent validity of this study. Individuals in the control group appeared more consistent in their performance on both measures than subjects with lateral epicondylitis, as evidenced by the larger standard deviation in the LE group, compared to the control group. This greater variability of test scores for the lateral epicondylitis group was consistent with the experience of the tester in clinical practice. This variability was probably not directly due to pain during testing, as any subject that described discomfort during the testing was excluded from data collection, and implies that the population of subjects with lateral epicondylitis may contain sub-groups with varying degrees of fine motor control. Much larger numbers of subjects would be required to test this possibility.

No significant effect for hand dominance was observed in this study. Some previous studies have demonstrated an effect for hand dominance on measures of fine finger dexterity (Chan, 2000; Kauranen & Vanharanta, 1996; Triggs, Calvanio, Levine, Heaton, & Heilman, 2000), while others studies have not (Ruff & Parker, 1993; Shahar, Kizony, and Nota, 1998). The main purpose of this study was not to determine the effect of hand dominance on fine motor control; however, I attempted to account for the possible effect of hand dominance by matching groups on this factor. It is possible that the sample size for this study (28 subjects) was too small to determine an effect for hand dominance. Lateral epicondylitis usually affects the dominant limb in approximately 76 percent of cases (Rotini, Fontana, Catamo, Noia, & Magnani, 2000). This study contained only 9 subjects out of 28 (32%) with lateral epicondylitis in their non-dominant limb. This small number of subjects with lateral

epicondylitis in their non-dominant hand also may have contributed to the difficulty in determining an effect of hand dominance, and the results should therefore be interpreted with caution. A larger sample of subjects with lateral epicondylitis in their non-dominant limb could be explored in future research, and may help clarify this question.

Subdividing the LE group, and comparing the acute LE group ($\leq$ 3 months) and chronic LE group (> 3 months), indicated that the time since injury did not affect fine motor control performance. Although it is recognized that the sample size was small for both groups (acute n = 13, chronic n = 15), this does raise some questions regarding the possible time course of the observed difficulty in fine motor control. It was initially suspected that persons with more chronic cases of LE would also have worse scores on tests of fine motor control; however this was not the case. The results from animal research by Barbe et al. (2003) suggests that multiple pathomechanisms may exist during the development of tendonitis, and that simultaneous pathophysiological changes may occur in the musculoskeletal system and neurological system adaptation (Barr & Barbe, 2002; Barbe et al., 2003; Moore, 2002). Further research with a larger sample size would be needed to confirm the current findings.

The design of this current study did not allow determination of whether the lateral epicondylitis preceded the observed deficits, or whether the deficits in fine motor control preceded the development of the lateral epicondylitis. Other painful soft tissue conditions have been shown to be associated with deficits in motor control but the

time course of development of these alterations in motor control is not known (Ferreira, P.H., Ferreira, M.L., & Hodges, 2004; Hodges & Moseley, 2003). One possibility is that subjects with decreased fine motor control alter their movement patterns and "overuse" their wrist and fingers during manual tasks, thus increasing the load on the tendon and predisposing the individual to development of tendonitis. In clinical practice, it has been noticed that there is an individual susceptibility to the development of tendonitis in workers that appear to be working under the same physical conditions. Alternately, multiple simultaneous changes may occur in both the musculoskeletal and neurological systems during the development of tendonitis, such as lateral epicondylitis, as suggested by Barr & Barbe (2003).

The mechanisms involved in the decrease in fine motor control in the subjects with lateral epicondylitis in this study are not known. The literature related to this area does, however, offer some possible theories. Experimental primate studies (Byl et al., 1996; Jenkins et al., 1990), and human studies with musicians (Byl et al., 2000; Elbert et al., 1998; Pantev et al., 2001), have demonstrated that reorganization of the cortical representation of the somatosensory cortex (Brodman's area 3b) can occur with repetitive use. These neuroplastic changes potentially may result in decreased fine motor control and increased load on the tendon structures, however, there is limited research in this area and further research to corroborate the findings of Byl and associates is needed. It is recognized that motor function is a result of complicated dynamic interaction between the sensory and motor systems, occurring at multiple spinal and supraspinal levels within the neurological system, and the

difficulties in fine motor control found in subjects with LE in this study does not explicitly mean that this is due to cortical remodelling (Lenz and Byl, 1999).

The effect of a chronically painful condition such as lateral epicondylitis may be a potential factor related to the observed deficits in fine motor control. Pain itself did not appear to be a factor during the testing, as only two subjects in the lateral epicondylitis group complained of discomfort during testing (these subjects were excluded). There may, however, be other behavioural responses to the pain associated with lateral epicondylitis. For example, it is possible that the LE subjects had learned, over an extended period that pain was associated with hand use, and this may have led to avoidance of certain activities and alterations in motor patterns (Heuts, Vlaeyen, Roelofs, de Bie, Aretz, van Weel, & van Schayck, 2004). Thus, even though the test itself was not painful, the fact that it involved use of the wrist and hand may encourage use of an abnormal, learned motor pattern. Chronic pain over an extended period can alter motor performance through altered sensory feedback and neural mechanisms such as peripheral or central sensitization, and reorganization within the central nervous system (Edwards, 1988; Flor, 2003; Gianberardino, 2003; Harris, 1999). Changes in motor performance also can accompany painful soft tissue injuries through "pain-adaptation", including increased recruitment of the antagonist muscle and decreased recruitment of the agonist muscle, and difficulty with synergistic muscle use (Graven-Nielsen, Svensson, & Arendt-Nielsen, 2000; Lund, Widmer & Stohler, 1991; Smeulders, Kreulen, & Bos, 2000; Sterling & Jull, 2001). Peripheral factors might also be involved in the impairment of fine motor control observed in this study. Eddema in the area of the tendon and surrounding structures may cause

increased pressure that may alter the accuracy of the sensory information provided by the proprioceptors, muscles spindles, and cutaneous receptors, thereby affecting the sensory feedback mechanism (Bjur, Alfredson, & Forsgren, 2005; Byl et al, 1996).

Limitations

It is recognized that the selection of the experimental group was a non-randomized convenience sample, as it includes subjects who attended for treatment rather than sampling the general population, which may threaten external validity. The results of this study can, therefore only be generalized to a population of similar characteristics.

A potential factor influencing the results of this study includes strength/endurance limitations. The Purdue Pegboard and the CMDT tests are short and non-strenuous tests of finger dexterity, and the weight of the pin or plastic block is well within the strength ability of the subjects with lateral epicondylitis. Fatigue is likely not a factor, as the trials are of short duration and adequate breaks were allowed between trials for recovery. Motivational factors are a potential factor in any test of human performance. The Purdue Pegboard and the CMDT tests are well standardized and structured as to the verbal instructions given to each subject; however, the response of subjects to the standardized instructions could vary. In the opinion of the tester, all subjects demonstrated competitive test performance when requested to complete the task.

Sampling bias was minimized through the use of age, gender, and dominance-matched control subjects. All LE subjects were screened using the inclusion and exclusion criteria to exclude possible neurological or medical causes for difficulty with motor control, and the diagnostic criteria were used to confirm the presence of lateral epicondylitis. Both control and LE subjects were referred for occupational injuries, however, the effect of occupation was not specifically controlled for in this study. This could be controlled for in future studies by testing subjects of a specific occupation which has been shown to have a high incidence of lateral epicondylitis.

The instruments used in this study are well standardized in terms of administration protocols to minimize rater and instrument bias. The use of alternate forms of measuring fine motor control improved confidence in the reliability of the instruments used, and offered concurrent validity. Rater bias was minimized by the use of objective scoring criteria; however, it is acknowledged that the examiner was not blinded to the subject group assignment, and this may possibly have influenced the subjects' performance. In future studies, the use of tape recorded instructions instead of verbal instructions with each client could help limit the effect of possibly giving a different presentation of instructions to each subject by the researcher. Another potential limitation of this study is that, for pragmatic reasons, data was collected from four clinical sites, two in Alberta (experimental subjects) and two in British Columbia (control subjects). In the LE group, 53% of the subjects were recruited from the CARD program at Millard Health in Edmonton, Alberta, and 47% of the subjects were recruited from the CARD program in Calgary, Alberta. In the control group, 74% of the subjects in the control group were recruited from Sun City

Physiotherapy, Kelowna, BC, and 26% were recruited from Columbia Health, Kelowna, BC. It is possible that the physical characteristics of the subjects attending each clinical site are somehow different and could influence their fine motor control ability.

CONCLUSIONS AND CLINICAL IMPLICATIONS

The purpose of this study was to determine whether difficulties in fine motor control existed in clients with lateral epicondylitis, compared to matched control subjects. Subjects with lateral epicondylitis in this study demonstrated a significant decrease in fine motor control ability when compared to age, and gender, and dominance matched control subjects (as measured by the scores of both the Purdue Pegboard Test and the Complete Manual Dexterity Test). While the results of this study are statistically significant, the PPT and CMDT are measures of impairment and it is not known how the observed deficits would affect the individual functionally. A study examining the relationship between and the degree of impairment and the level of functional disability would be helpful.

The One-handed placement portion of the PPT, and the One-handed turning and placing portion of the CMDT appeared equally sensitive in detecting differences in fine motor control ability in these subjects with lateral epicondylitis. The PPT, however, may be the more practical test for use with patients, as it is less expensive and time consuming for the practicing clinician. The use of these common clinical assessment tools allows a practical method for clinicians working with patients with

lateral epicondylitis to similarly identify if there are problems with motor control. This factor could then be addressed in the patient's rehabilitation program.

The results of this study indicate there was a difference in fine motor control between the LE group and matched control subjects, but do not explain or imply causation for the decrease in fine motor control. This study does, perhaps, add to the list of possible factors related to lateral epicondylitis, and may offer some explanation of why some subjects complain of clumsiness in their affected limb. Deficits in fine motor control also may provide some explanation for why a biomechanical treatment approach is not effective in all cases of lateral epicondylitis. Authors in this area have recommended that the possibility of dysfunction in neuromuscular control should be considered in assessment and treatment of repetitive strain disorders (Barr, Barbe, & Clark, 2004; N. Byl et al., 2000; McKenzie, Nagarajan, Roberts, Merzenich, & Byl, 2003). Byl (2004) recommended that adjunctive treatment could include sensorimotor retraining exercises aimed at restoring motor control, rather than solely treating local muscle-tendon signs and symptoms. It is not known as yet, however, if improvements in fine motor control will have a direct impact on the resolution of lateral epicondylitis, and this warrants further research. Measurement of fine motor control may help select subjects with lateral epicondylitis who could benefit from specific motor control training. Comparison of the outcomes of such training, compared with standard, or no, treatment, would help determine: a) whether such treatment helps improve fine motor control; and b) whether such treatment improves overall treatment of lateral epicondylitis.

There is the potential of several future research studies in this area. Though the PPT and CMDT appear to be valid measures for assessing within-subject treatment effectiveness, new normative studies for the measures used in this study are needed to enable clinicians to compare the scores they obtain in a clinical setting to normative data for subjects with different ages, genders and occupations. A prospective, longitudinal study comparing the cortical representation of LE subjects who have clinical deficits in fine motor control during the recovery of their lateral epicondylitis may help in the understanding of whether cortical reorganization is a possible pathomechanisms in this population. Further, if an impairment in fine motor control is thought to be at least partially responsible for the peripheral pathological changes noted in lateral epicondylitis, it would be expected that an improvement in test scores for fine motor control would accompany resolution of the patient's signs and symptoms, and a longitudinal study testing the fine motor control ability of patients attending treatment for lateral epicondylitis may be helpful. Alternatively, to help examine whether workers of with poor motor control are more susceptible to the development of lateral epicondylitis, the fine motor control performance of workers in industries with a high incidence rate of LE could be tested over time to see if deficits in fine motor control help predict the incidence of lateral epicondylitis.

In conclusion, the exact mechanisms or the functional significance of the observed deficit in fine motor control is this study are unknown. It is likely that there are many factors involved in the complex presentation of patients with recalcitrant upper extremity tenopathies, and further research in this area is needed.

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Appendix A

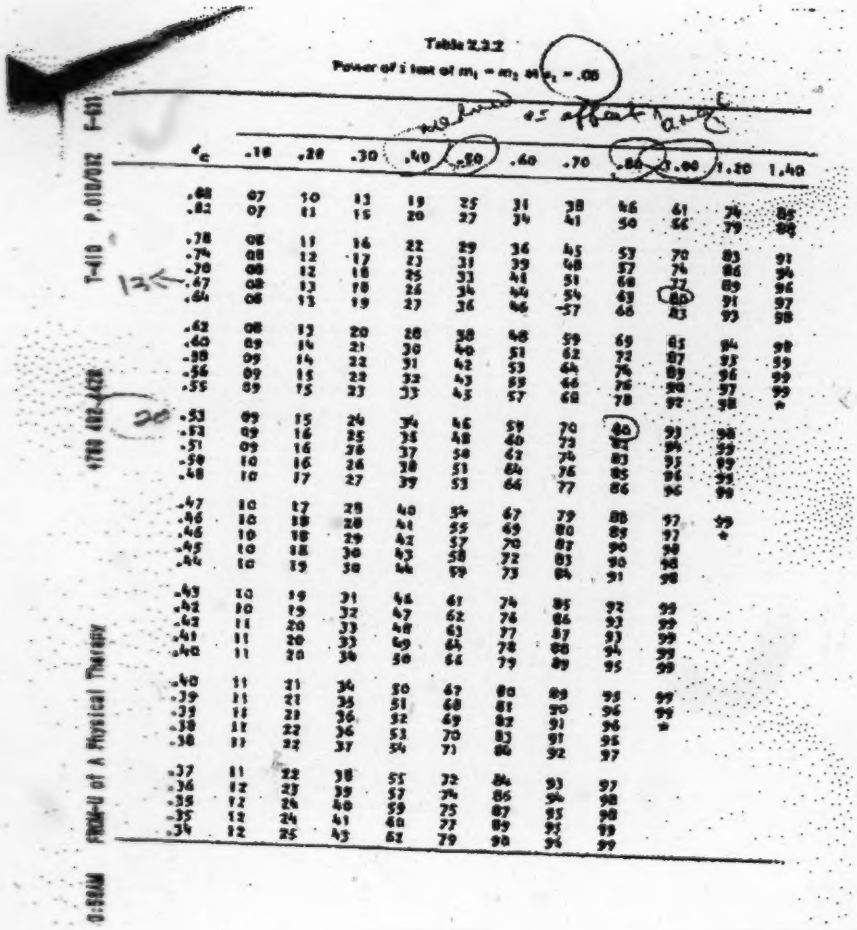

Table 2.3.2

Power of 1 test of $m_1 = m_2$ at $\alpha = .05$

d_c	.10	.20	.30	.40	.50	.60	.70	.80	.90	1.00	1.20	1.40
.68	07	10	13	19	25	31	38	46	61	74	86
.82	07	11	15	20	27	34	41	50	66	79	90
.78	08	11	16	22	29	36	45	57	70	83	91
.74	08	12	17	23	31	39	48	57	74	86	94
.70	08	12	18	25	33	41	51	60	77	89	96
.67	08	13	18	26	34	44	54	63	80	91	97
.64	08	13	19	27	36	46	57	66	83	93	98
.62	08	13	20	28	38	48	59	69	85	94	98
.60	09	14	21	30	40	51	62	72	87	95	99
.58	09	14	22	31	42	53	64	74	89	96	99
.56	09	15	22	32	43	55	66	76	90	97	99
.55	09	15	23	33	45	57	68	78	92	98	*
.53	09	15	24	34	46	59	70	80	93	98
.52	09	16	25	35	48	60	72	82	94	99
.51	09	16	26	37	50	62	74	83	95	99
.50	10	16	26	38	51	64	76	85	96	99
.48	10	17	27	39	53	66	77	86	96	99
.47	10	17	28	40	54	67	79	88	97	99
.46	10	18	28	41	55	69	80	89	97	*
.46	10	18	29	42	57	70	82	90	98	
.45	10	18	30	43	58	72	83	90	98	
.44	10	19	30	44	59	73	84	91	98	
.43	10	19	31	44	61	74	85	92	99	
.42	10	19	32	47	62	76	86	93	99	
.42	11	20	33	48	63	77	87	93	99	
.41	11	20	33	49	64	78	88	94	99	
.40	11	20	34	50	64	79	89	95	99	
.40	11	21	34	50	67	80	89	95	99	
.39	11	21	35	51	68	81	90	96	*	
.39	11	21	36	52	69	82	91	96		
.38	11	22	36	53	70	83	91	96		
.38	11	22	37	54	71	84	92	97		
.37	11	22	38	55	72	84	93	97		
.36	12	23	39	57	74	86	94	98		
.35	12	24	40	59	75	87	95	98		
.35	12	24	41	60	77	89	95	99		
.34	12	25	43	62	79	90	96	99		

Appendix B

Matching of Experimental and Control Pairs - genders matched across each pair

	Experimental		Control	
Pair	**Age**	**Gender**	**Affected Limb**	**Age**
1	30	M	Dom	30
2	30	M	Dom	30
3	32	M	Dom	33
4	33	M	Dom	34
5	34	M	Dom	34
6	40	M	Dom	42
7	41	M	Dom	42
8	41	M	Dom	43
9	42	M	Dom	44
10	45	M	Non-dom	44
11	46	M	Non-dom	48
12	50	M	Dom	48
13	34	F	Dom	32
14	37	F	Dom	33
15	39	F	Non-dom	39

Experimental			Control	
Age	**Gender**		**Affected Limb**	**Age**
16	41	F	Non-dom	41
17	41	F	Dom	43
18	42	F	Dom	45
19	44	F	Dom	46
20	45	F	Dom	45
21	45	F	Non-dom	46
22	46	F	Dom	46
23	46	F	Non-dom	47
24	47	F	Dom	48
25	50	F	Non-dom	50
26	50	F	Non-dom	49
27	50	F	Dom	50
28	53	F	Dom	52

Appendix C

Development and Testing of the Purdue Pegboard Test and the Complete Manual Dexterity Test

Purdue Pegboard

The PPT was initially developed in 1948, by Dr. Joseph Tiffen at the Purdue University, for the testing of fine and gross motor control of industrial applicants (Tiffen, 1948). The PPT has since been used in a variety of clinical settings to evaluate and document changes in fine motor control (Pinkowski, 2002). Examples of different uses of the test includes: the evaluation of fine motor control in children (Mathiowetz et al., 1986) and older adults (Desrosiers et al., 1995), musicians (Genc, 2002), and persons receiving vocational rehabilitation (Hamm & Curtis, 1980), to measure the functional consequences of short term arm and hand vibration (Malchaire, Rodriguez Diaz, Piette, Goncalves Amaral, & de Schaetzen, 1998), following traumatic hand or finger disabilities (Pennathur, 1999; Shahar, 1998), in the evaluation of wrist orthosis in patients with rheumatoid arthritis (Stern, Ytterberg, Krug, & Mahowald, 1996), to assess upper extremity function in hemiplegic subjects (Smutok, 1989), as an evaluation tool with multiple sclerosis patients (Gallus & Mathiowetz, 2003), as a predictor of the presence of cerebral lesions (Costa, Vaughan, Levita, & Farber, 1963), to measure the functional consequences of lead poisioning (Stewart et al., 1999), to evaluate the effectiveness of botulinum toxin injections in patients with spastic upper extremities (Hurvitz, Conti, & Brown, 2003), and in the evaluation of entrants to dental hygiene training (Waldman, 1995). The

PPT has also been used to evaluate of the validity of other tests of manual dexterity (Maiden, 1997).

In the original studies of 434 college men and women, test-retest reliability for three trial scores of one-handed placement portion was measured at .82 using the left hand, and .84 using the right hand (Tiffen, 1948). The board was slightly modified in 1960 and test-retest correlations were determined for the new Purdue Pegboard (Model 32020) in 1965, and revealed reliability coefficients of .84 for the left hand to .86 for the right hand (Instructions and Normative Data for Model 32020 Purdue Pegboard, Lafayette Instrument). Using the arithmetic sum of three trials was chosen for this study as the reliability correlations were obviously greater than using only one trial.

Test – Retest Reliability Studies of Original PPT*

One Handed Placement	**n =**	**One trial**	**Sum of three trials ***
Right hand	434	.63	.84
Left hand	434	.60	.82

* means three trial reliability estimated by means of the Spearman – Brown prophecy formula

Tiffen, J. (1948). "The Purdue Pegboard: Norms and studies of reliability and validity." J Applied Psych **32**: 234.

Test – Retest Reliability of the 32020 Purdue Pegboard

One Handed Placement	Industrial Education College Students n = 60		Professional and Editorial Personnel n = 28	
	1 Trial	3 Trials *	1 Trial	3 Trials *
Right hand	.67	.86	.68	.86
Left hand	.66	.85	.65	.84

* means three trial reliability estimated by means of the Spearman – Brown prophecy formula

Instructions and Normative Data for Model 32020 Purdue Pegboard. LaFayette, IN, Lafayette Instrument

The validity of the PPT was tested by comparing test scores of 760 airmen entering a Air Force Technical School to their actual performance on a specific job for which the test was used for selection (Fleishman & Ellison, 1962). The results of the PPT were compared to eleven other apparatus and nine printed fine manipulative tests, and considered a valid test of fine finger dexterity. The validity of the PPT has been confirmed using other populations, such as light machine operators, assemblers, seed analysts, packers, and high school shop trainees (Instructions and Normative Data for Model 32020 Purdue Pegboard, Lafayette Instrument Company). The PPT is a well recognized test in the fields of vocational rehabilitation and hand therapy.

Complete Manual Dexterity Test

The Minnesota Rate of Manipulation Test (MRMT) was first developed in 1946, and like the Purdue, measures a person's ability to rapidly manipulate objects. There were some minor changes made to the MRMT in the 1969, and the newer version was called the Complete Minnesota Dexterity Test (CMDT). This test was first developed for screening of industrial applicants, however, it has since been commonly used in occupational therapy departments to assess dexterity disability and for vocational assessment (Desrosiers et al., 1997). The reliability and validity of the CMDT has been tested with a variety of populations (Desrosiers et al., 1997). The One-handed Turning and Placing portion of the CMDT tests the time recorded to turn, move and place 60 disc shaped objects in a prescribed space on the board. This subtest was chosen as it is the most sensitive for assessing finger dexterity as it involves manipulation of the object with the fingertips, and is well standardized.The two-trial reliability of the One Handed Turning and Placing subtest was reported as .95, and the four-trial reliability as .98 (n=212) (The Complete Minnesota Dexterity Test: Examiner' Manual, 1969, Lafayette Instrument). Four trials will therefore be used for research purposes in this study, after one trial is given for practice.

Test – Retest Reliability of the Complete Minnesota Dexterity Test

Subtest	**Two – Trial Reliability n = 212**	**Four – Trial Reliability n = 212**
Placing	.87	.93
Turning	.91	.95
One-Handed Placing and Turning	.95	.98
Two-handed Placing and Turning	.94	.97

The Complete Minnesota Dexterity Test: Examiner' Manual, 1969, Lafayette Instrument).

Validity of the CMDT was reported to be tested by Jurgensen in 1943, by correlating independent criterion performance ratings of 60 pulp workers by their supervisors to the results of the Complete Minnesota Dexterity Test (The Complete Minnesota Dexterity Test: Examiner' Manual, 1969, Lafayette Instrument).The validity coefficient for the One-handed Turning and Placing test portion of the CMDT was reported to be .67. The Minnesota test was further validated against another test of motor skill; the Pennsylvania Bi-Manual Worksample test (n=473, r=.40) (The Complete Minnesota Dexterity Test: Examiner' Manual, 1969, Lafayette Instrument).